OK TO ENTER: /C.Y.W./ 06/04/2009

Amendments to the Specification

Please replace the paragraph beginning at page 1, line 1, with the following amended paragraph:

TITLE: Detection of Pathogenic Polypeptides using an Epitope Protection Assay ~~and Method for Detecting Protein Conformations~~

Please replace the paragraphs beginning at page 6, line 1 to page 7, line 20, with the following amended paragraphs:

Alzheimer's Disease

AD is a common dementing (disordered memory and cognition) neurodegenerative disease associated with brain accumulation of extracellular plaques composed predominantly of the Abeta (1–40), Abeta (1-42) and Abeta (1–43) peptides, all of which are proteolytic products of APP (reviewed in 4). In addition, neurofibrillary tangles, composed principally of abnormally phosphorylated tau protein (a neuronal microtubule-associated protein), accumulate intracellularly in dying neurons (4). Familial forms of AD can be caused by mutations in the APP gene, or in the presenilin 1 or 2 genes (~~www.websiteformutations.com~~), the protein products of which are implicated in the processing of APP to Abeta. Apolipoprotein E allelic variants also influence the age at onset of both sporadic and familial forms of AD (reviewed in 5). Abeta has been detected in the blood and CSF of AD patients and in normal controls (6). Abeta is also present in vascular and plaque amyloid filaments in trisomy 21 (Down's syndrome), hereditary cerebral hemorrhage with amyloidosis (HCHWA)-Dutch type, and normal brain aging (Mori, H et al. JBC (1992) 267: 17082-86). Tau and phosphorylated tau have been detected in the cerebral spinal fluid (CSF) of AD patients and patients with other neurological diseases (7; reviewed in 8).